ANGINA PECTORIS

MEDICAL

TRANSACTIONS,

COLLEGE of PHYSICIANS

LONDON.

VOLUME THE SECOND.

THE SECOND EDITION.



LONDON:

§ I. *Some Account of a Disorder of the Breast. By* WILLIAM HEBERDEN, *M. D. F. R. S.*

Read at the COLLEGE, July 21, 1768.

THERE is a disorder of the breast, marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, of which I do not recollect any mention among medical authors. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called Angina pectoris.

Those, who are afflicted with it, are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would

would take their life away, if it were to increase or to continue: the moment they stand still, all this uneasiness vanishes. In all other respects, the patients are, at the beginning of this disorder, perfectly well, and in particular have no shortness of breath, from which it is totally different.

After it has continued some months, it will not cease so instantaneously upon standing still; and it will come on, not only when the persons are walking, but when they are lying down, and oblige them to rise up out of their beds every night for many months together: and in one or two very inveterate cases it has been brought on by the motion of a horse, or a carriage, and even by swallowing, coughing, going to stool, or speaking, or by any disturbance of mind. I have heard one, and only one person, say, that he had known

Heberden's original description of Angina Pectoris

SCRIPT OF

ANGINA PECTORIS

A MOTION PICTURE

WRITTEN AND DIRECTED

BY

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FOREWORD

The motion picture Angina Pectoris and this booklet are intended to present, in easily absorbable form, a Clinic on Angina Pectoris The importance of the clinic method of presenting medical information is of recognized value, but the difficulties of assembling patients and material limits its use Motion pictures correct some of the difficulties associated with "Dry Clinics," and they have become a valuable method of disseminating medical knowledge Since a motion picture is portable, it makes a Clinic available to any one, anywhere, any time, it eliminates the difficulties and dangers of assembling patients on time for each presentation, it makes it possible to demonstrate a wealth of material collected, if necessary, from all parts of the globe, furthermore, errors, excess words, illegible charts, etc., can be eliminated in the cutting room As a result the motion picture method of presenting data is not only interesting but it makes it possible for the observer to absorb a considerable amount of knowledge with a minimum amount of concentration, effort and reading The finished product may result, however, in such a wealth of material being presented in so short a period of time that the observer is unable to absorb it at one sitting and must either see the picture again or have a means of referring to selected portions of it at his leisure This illustrated booklet was prepared to supply this need

It is hoped that the booklet, combined with the motion picture, will aid the student in obtaining a sound understanding of the important facts about Angina Pectoris and will serve as a useful review to those physicians whose busy lives make it impossible for them to read all the publications necessary to obtain this data The aspects presented are based primarily on work done at the Beth Israel Hospital of Boston They represent not theories or animal experiments but facts demonstrable by objective studies in patients with angina pectoris

I wish to acknowledge the valuable assistance of the following who worked without thought of time or remuneration Dr Elliot L Sagall who helped in the preparation of the sets, Mr James Brewster of the Harvard Film Service for his technical skill and wise counsel, Miss Isabelle Levi for her assistance in preparing the script, and Mr Howard D Brewer, president of Brewer & Company, Inc, for his interest in medical research and teaching and whose generous grant made this presentation possible

JOSEPH E F. RISEMAN, M D
Boston, Mass

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Part 1

Clinical Characteristics of Angina Pectoris

PART 3

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PART I

CLINICAL CHARACTERISTICS OF ANGINA PECTORIS

In 1768, William Heberden presented before the College of Physicians, in London, a treatise entitled "Some Account of a Disorder of the Breast (Frontispiece) This begins as follows *"There is a disorder of the breast, marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, of which I do not recollect any mention among medical authors The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called Angina Pectoris."*

The patient presented in plates 1 and 2 is 65 years of age She has had angina pectoris for six years Like most patients with this condition, she is perfectly comfortable between attacks (Plate 1, page 25) On exertion, however, and occasionally on emotion or even while at rest or during sleep, she may develop the characteristic symptoms which we call angina pectoris (Plate 2, page 25)

As will be shown, attacks of angina can be precipitated by having patients repeatedly mount a two-step staircase. During such episodes this patient illustrates dramatically the typical clinical characteristics of this condition

First—the attacks are obviously uncomfortable (Plate 2, page 25).
Second—the discomfort is located in the anterior chest and inner aspects of the arms
Third—the attacks are most commonly precipitated by exertion
Fourth—they are sudden in onset The pain in this patient did not develop until about 10 seconds before she was forced to stop
Fifth—the attacks are short in duration

The attack photographed corresponds closely to the description given in most textbooks, in fact this is the reason that this particular patient was selected The severity of the pain in this individual and the severity of this patient's reaction to attacks, however, are distinctly unusual In the attack photographed pain was precipitated by 21 trips over the two step staircase performed in 1 minute and 38 seconds The attack lasted 1 minute and 42 seconds

The more common appearance of the attacks as they occur in over 95% of patients is shown in figure 1



(Figure 1)

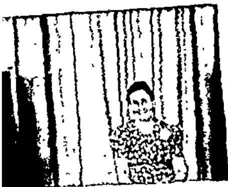


Plate 1 —Patient B. K.



Plate 2 —"Textbook" attack of angina pectoris



Plate 3 —Distribution of pain in angina.

DIAGNOSTIC CRITERIA

1. S
2. A ST
3. V FORT
4. E UCES
5. SHORT DURATION

Plate 4 —Clinical criteria diagnostic of angina pectoris.

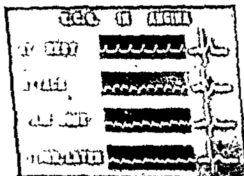


Plate 5 —Continuous electrocardiograms during angina on effort

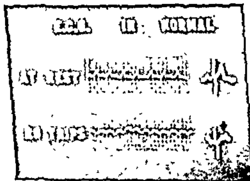


Plate 6 —Continuous electrocardiograms of normal during exercise

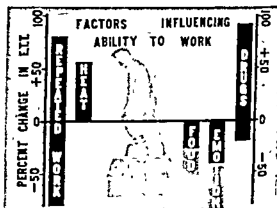
It is important to realize that our knowledge of angina pectoris rests almost entirely upon what patients are able to describe to us about their sensations. Furthermore, so far as we know, this condition exists only in human beings. Therefore, **it is necessary to study angina pectoris not in laboratory animals but in patients.**



(Figure 2)

The fact that most patients develop attacks on exertion makes it possible to study angina pectoris objectively in the laboratory. Such studies have been carried out since 1932 at the Beth Israel Hospital of Boston. As part of this work, attacks have been precipitated by having the patient exercise in the presence of a physician (fig 2). These studies have been carried out in a room equipped with a refrigerating unit to maintain a constant temperature of 45 to 55 degrees.

This cold room is necessary, for when the various factors influencing the amount of work which can be done before developing an attack were measured, it was soon found that some patients with angina can exercise almost indefinitely at room temperature, whereas, they develop attacks rapidly on exercise in the cold (fig 3). It was also found that a heavy meal will decrease the amount of work which can be done before pain. Excitement or emotion also increases the ease of precipitating attacks. If exercise is repeated or continued, we find that in some patients one attack builds up a slight immunity, while in others the first attack predisposes to a second episode. Similarly, medication may influence the ease of precipitating pain. As a result of these studies, the Standardized Exercise Tolerance Test was developed. This test reproduces in the laboratory the conditions responsible for the majority of attacks in daily life and makes it possible to study angina pectoris objectively without relying on the patient's observations for our basic knowledge.



(Figure 3)

The standardized conditions used in these tests are as follows (fig 4). The temperature of the room is maintained between 45 and 55



Plate 1—Patient B. K.



Plate 2—"Textbook" attack of angina pectoris



Plate 3—[illegible] of pain in angina

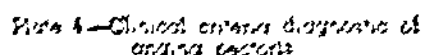


Plate 4—Clinical criteria diagnostic of angina pectoris

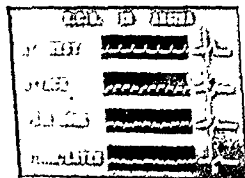


Plate 5—[illegible] electrocardiograms during angina [illegible]

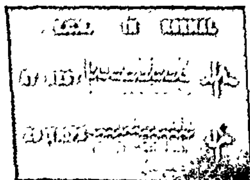


Plate 6—Continuous electrocardiograms of normal during exercise

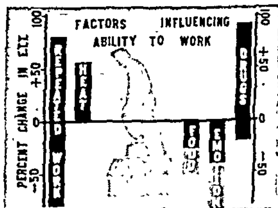
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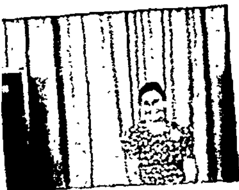


Plate 1.—Patient B. K.



Plate 2.—"Textbook" attack of angina pectoris.



Plate 3.—Distribution of pain in angina.

DIAGNOSTIC CRITERIA

1. S ... SET
2. A ... ST
3. V ... FORT
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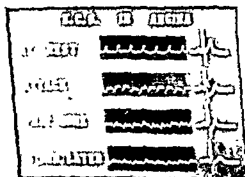


Plate 5.—Continuous electrocardiograms during angina on effort.

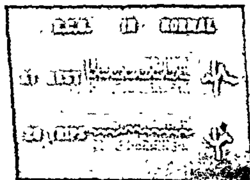
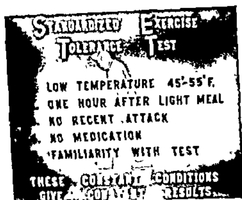


Plate 6.—Continuous electrocardiograms of normal during exercise.



(Figure 4)

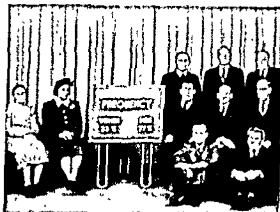
degrees Fahrenheit The exercise is carried out at least one or two hours after a light meal, and the patient rests for one-half hour after coming to the laboratory. No medication is given for at least 12 hours before the test Only one measurement is carried out on any given day, and no work is performed if the patient has experienced an attack on the way into the hospital, except in those individuals who always experience many attacks every day Since excitement frequently induces attacks, it is necessary to minimize the emotional factor, familiarity with the test brings this about

The attack shown in patient R S (fig 1, page 23) does not correspond closely to the usual textbook description but it is absolutely typical of the usual attacks as they occur in daily life in the vast majority of patients The patient shows no expression of severe discomfort, although he prefers to remain perfectly quiet. There is no fear of impending death and no pallor, flushing, fainting, perspiration or vomiting as has been described by some authors Although the patient feels that the attack has persisted for some minutes, actual measurement shows that it was about 38 seconds in duration

The characteristic regions in which patients feel the pain are as follows (Plate 3, page 25) The anterior chest is always involved in an area limited by the anterior axillary lines on the sides, the suprasternal notch above, and the epigastrium below The region beneath the sternum is the most common location Radiation of the pain occurs in approximately 60% of patients The most common region to which the discomfort travels is the left arm, along the inner aspect of the biceps, the elbow, the forearm, the wrist, and the little and ring fingers In some patients it is referred to similar locations in the right arm The upper back, in the region of the spine, is commonly involved, although the pain here is not disturbing to the patient Rarely, it radiates to the neck or to other regions such as the lower jaw

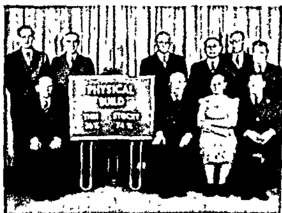
It has been said that the usual patient with angina is short and stocky, of the so-called vascular build, and that they show abnormalities in the electrocardiogram, blood pressure or heart size The frequency of the different physical characteristics as actually found in a group of 100 of these patients is as follows

FREQUENCY Heberden stated· *"I have seen nearly a hundred people under this disorder, of which number there have been three women."* More recent studies show that it is 3 to 4 times as common in men as in women (fig 5)



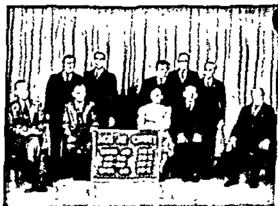
(Figure 5)

PHYSICAL BUILD While it is true that the majority of *patients are stocky, obese, or* "vascular", there are striking exceptions to this rule It is obvious that the build of the patient is of no aid in establishing the diagnosis (fig 6)



(Figure 6)

AGE AT ONSET Angina pectoris *may occur at any age,* but it is not common in patients under 40 About 80% of patients are between 45 and 65 years of age when the symptoms first become evident (fig 7)



(Figure 7)



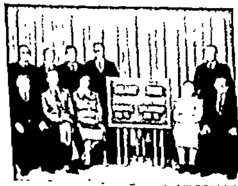
(Figure 8)

BLOOD PRESSURE. Since one-half of the patients have normal blood pressures it is obvious that hypertension is not an important factor in the diagnosis of angina pectoris (fig 8)

ELECTROCARDIOGRAM Similarly, only one-half of the patients show evidence of heart disease in the standard electrocardiogram This is somewhat surprising in view of the fact that these patients all have heart disease, but the heart has many areas which are silent electrocardiographically (fig 9)

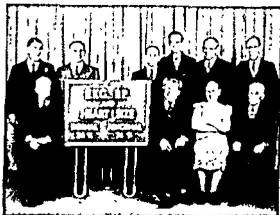


(Figure 9)



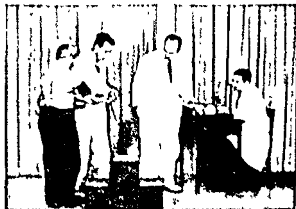
(Figure 10)

HEART SIZE Only one-fourth of patients with angina pectoris show cardiac enlargement, even when the measurements are made by X-Ray. This is in sharp contrast to the findings in other forms of heart disease such as congestive failure (fig 10)

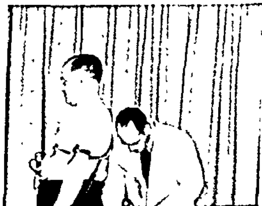

(Figure 11)

ELECTROCARDIOGRAM, BLOOD PRESSURE, and HEART SIZE. One-fourth of the patients with angina show no abnormalities in electrocardiogram, *blood pressure* or heart size, even though the disease may be advanced and the symptoms severe (fig. 11). **It is important to realize that abnormalities in electrocardiogram, blood pressure or heart size are evidence of cardiac damage and are not evidence of heart pain.**

Since the attacks come suddenly, without warning, and are of short duration, it is obvious that opportunities to make measurements during spontaneous attacks are rare The heart rate and the systolic blood pressure can be obtained during exercise quite readily by a special technique (fig 12) Such observations show no constant relationship between the heart rate or the height of the blood pressure and the onset or precipitation of the attack


(Figure 12)

A few observers have been fortunate enough to obtain electrocardiograms during spontaneous attacks The short duration of the pain, and the time necessary for the technical *procedure* of taking such tracings, suggests that these electrocardiograms were obtained toward the end or even after the acute discomfort The Standardized Exercise Tolerance Test makes it possible to take electrocardiograms continuously before, during and after the attack (fig 13)


(Figure 13)

ELECTROCARDIOGRAM DURING ATTACKS OF ANGINA PECTORIS

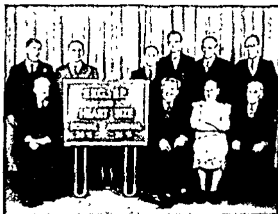
The electrocardiogram taken continuously during the precipitation of an attack practically always shows characteristic changes, especially in the precordial lead. (Plate 5, page 25). These changes begin shortly after the onset of exercise and increase slowly, reaching a maximum at the time when the patient is forced to stop because of pain. The changes consist of a deviation, usually a depression, of the S-T segment as can be seen by comparison with the tracings taken at rest. Thereafter, they either subside rapidly or show further progression, especially in the height of the T waves. While changes in the T waves may occur during the attack, they are more common during recovery from the pain and the exertion.

The marked S-T changes found in patients with angina after exertion are not common in normals, but they do occur (Plate 6, page 25). Changes in the T waves, however, are quite frequent. In our experience the electrocardiogram after exercise is of little practical value in the diagnosis of angina pectoris.



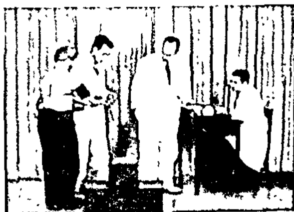
(Figure 14)

It has also been found that patients with angina pectoris may show these S-T and T changes when breathing a low concentration of oxygen (figs. 14 and 19). Many patients fail to show such changes, however, while an occasional normal individual may show abnormalities which are indistinguishable from those observed in patients with angina. It is obvious, therefore, that such changes are of little diagnostic value.

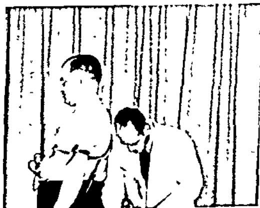

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(Figure 13)

Part 2

Physiology of Angina Pectoris

Obviously, there is little in the physical examination, X-Ray, blood pressure, or electrocardiogram which is of value in the diagnosis of angina pectoris By observation of patients during attacks, the important diagnostic characteristics were found to be (Plate 4, page 25)

1 The attacks are **sudden** in onset with little or *no* premonitory symptoms.

2 The discomfort is in the **anterior** chest Radiation of the pain is important if it occurs, but it may be absent

3. The discomfort is **vague** in character and difficult for the patient to describe

4 **Exertion** is the most common precipitating cause, *although it may also occur on emotion*

5 The attacks are **short** in duration According to the patient they rarely last longer than 15 to 20 minutes

Practically all patients show all five clinical characteristics Difficulties in diagnosis arise mainly because of the inability to obtain a clear cut description of the patient's symptoms Under such conditions, observation of the patient during a typical attack may be of considerable value in diagnosis

Heberden's classic description includes the five characteristics which recent experience has shown to be of diagnostic value (frontispiece)

"Those, who are afflicted with it, are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue: the moment they stand still, all this uneasiness vanishes."

PART 2

PHYSIOLOGY OF ANGINA PECTORIS

There is considerable evidence in the literature that attacks of angina pectoris occur when the heart muscle receives an insufficient amount of oxygen or blood or both This anoxemia or ischemia theory is consistent with more of the known clinical facts concerning angina pectoris than is any other concept Furthermore, it makes many characteristics of the disease more understandable There is considerable experimental evidence in favor of this theory

Lewis showed that muscular exercise, in the absence of an adequate blood supply, causes pain in the muscles used We can occlude the blood supply to the arm of a normal healthy subject by inflating a blood pressure cuff to a level greater than that of his arterial blood pressure (fig 15) If the muscles of the forearm are then exercised, the available oxygen dissolved in the tissues is soon used up, and the subject experiences first fatigue, then pain in those muscles involved This pain rapidly increases in severity and continues even though exercise is stopped As soon as the tourniquet is released however, fresh blood enters the arm and the pain disappears immediately



(Figure 15)

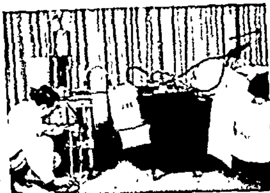
The clinical counterpart of this experiment is seen in patients who have arteriosclerosis involving the arteries of the heart and also those of the legs On exercise, such patients may develop not only angina pectoris but also pain in the calf muscles (fig 16) This condition called intermittent claudication' is fairly common among elderly individuals even in the absence of angina pectoris



(Figure 16)

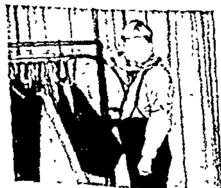
changes are associated with an insufficient supply of oxygen to the heart muscle. Their occurrence may be of value in establishing the diagnosis of myocardial infarction.

Another line of evidence in favor of the anoxemia theory was presented by Rothschild and Kissin in 1932 who showed that attacks of angina pectoris could be precipitated in some patients at rest by having them breathe low concentrations of oxygen (fig 19). The apparatus consists of a spirometer, such as an ordinary basal metabolism machine, connected in series with a reservoir of about 20 liters, and a motor blower to insure circulation. As a result of this re-breathing, the oxygen content, as determined by analysis, is decreased to as low as 8 or 10%. Under these conditions, some but not all patients with angina develop pain.



(Figure 19)

Electrocardiograms taken during such experiments (Plate 8, page 41) may show changes in the S-T segment and the T wave identical with those observed in the same patient during spontaneous attacks of angina or during attacks induced by exercise. These changes are similar to those found in some patients with coronary occlusion. Such studies indicate that attacks of angina are associated with myocardial anoxia regardless of whether they are produced spontaneously, by exercise, or by generalized anoxemia.

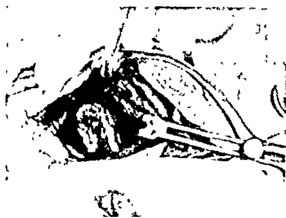


(Figure 20)

The opposite of this type of experiment has been performed by Riseman and Brown in 1939 (fig 20). Their investigations show that exercise while breathing pure oxygen enables patients with angina to do more work without pain than is possible under the usual standardized conditions, even though handicapped by the apparatus.

The presence of arteriosclerosis in the legs is indicated by the absence of palpable pulsations of the dorsalis-pedis and the posterior-tibial arteries and also by the relatively cold temperature of the feet This can be demonstrated by the sense of touch More accurate measurements with a dermatherm show that the temperature of the great toe is considerably less that that of other parts of the body, such as the thumb (fig 17)


(Figure 17)


(Figure 18)

In 1930, Sutton and Lueth showed experimentally the relationship of anginal pain to narrowing of the coronary arteries In these experiments an animal was anesthetized, the heart was exposed, and a ligature passed about one of the coronary arteries (fig 18) After recovering from this operation, they could occlude the artery at will by gentle traction This constriction caused discomfort in the chest and left paw

The clinical condition "coronary occlusion" simulates this experiment In this condition patients suddenly develop pain in the chest which is similar to their anginal attacks but is much more severe and continues for several hours Post-mortem examination of such individuals usually reveals that one of the major arteries of the heart has become completely occluded so that the cardiac circulation is restricted similarly to the artificial restriction in the experimental animal (see part 3)

Electrocardiograms taken during experimental coronary occlusion in dogs or coronary thrombosis in man (Plate 7, page 41) may show S-T segment and T wave changes similar in character but more pronounced than those seen in attacks of angina pectoris These

quate blood flow, or regurgitation of such a degree that during diastole a portion of blood flows back into the heart rather than through the coronary vessels

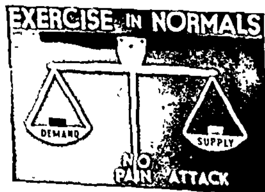
Abnormal rhythms, especially if the rate is rapid, may result in poor circulation Under such conditions, the increased work of the heart together with the poor blood flow, may result in myocardial anoxia and angina pectoris

In anemia, the decreased number of red blood corpuscles results in a decreased supply of oxygen to the heart In addition, the heart has to work harder to supply the necessary oxygen As a result, many elderly patients with anemia experience heart pain

In exophthalmic goiter, the tissues of the body, including the heart, require an increased amount of oxygen and the heart beats faster As a result, a few patients develop angina

Similarly, some patients with congestive failure, although complaining primarily of dyspnea, may experience a feeling of pressure, choking, squeezing or pain in the anterior chest or arms.

Patients with emphysema or asthma also complain of symptoms simulating angina pectoris In these individuals the abnormality of the lungs prevents adequate oxygenation of blood when the patient exercises



(Figure 22)

The mechanism of angina pectoris can be summarized and illustrated by the Law of Supply and Demand as it applies to the coronary circulation.

In the normal individual and in most patients with angina pectoris the **Supply** of blood through the coronary vessels is adequate for the needs or **Demands** of the myocardium when the subject is at rest (fig 22)

The theory of referred pain as presented by Head in 1893 explains why patients, who exercise with their legs, experience pain in the anterior chest and inner aspects of the arms

The usual distribution of pain in angina pectoris (Plate 9, page 41) is innervated, in the chest, by the eighth cervical, the first second, third, fourth, and fifth thoracic nerves The sensory innervation of the inner aspect of the arm is derived from the eighth cervical, the first and second thoracic nerves

The anatomic nerve supply to the heart is well known An actual dissection of these nerves shows that it is a most complex network (fig 21) *The cardiac nerve supply consists of fibers from* the vagus, which unite with the cervical sympathetic, and the thoracic sympathetic chain and ganglia (plate 10, page 41) Functionally, the sympathetic nerves connect with the ganglia of the eighth cervical, the first, second, third, fourth and fifth *thoracic nerves Impulses which arise* in the heart travel up the sympathetic nerves to the nerve roots in the dorsal ganglia and spinal cord The sensation of pain set up by these impulses is felt not in the *organ actually involved, but in the areas innervated by the* peripheral nerves arising in the same segments of the spinal cord (Plate 11, page 41)



(Figure 21)

Thus, the sensations set up in the heart by anoxemia are experienced as pain in the anterior chest and inner aspects of the arms

The anoxemia-ischemia theory helps considerably in understanding why angina pectoris is associated with certain diseases

For example, it is most commonly found in elderly individuals of the arteriosclerosis age group

Syphilis of the *aorta and mouths of the coronary arteries may* also be a cause of inadequate coronary circulation

Similarly, some patients with disease of the aortic valves may complain of angina pectoris In these patients, examination of the valves may reveal a degree of stenosis so great as to prevent an ade-

quate blood flow, or regurgitation of such a degree that during diastole a portion of blood flows back into the heart rather than through the coronary vessels.

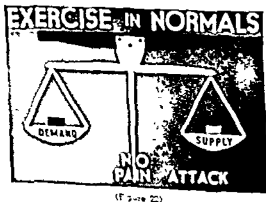
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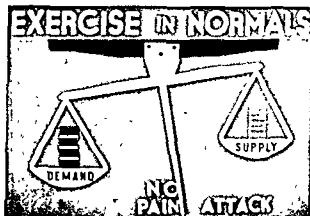


(Figure 22)

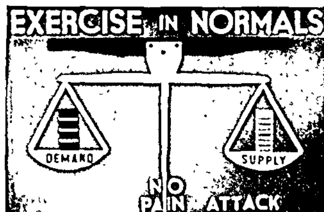
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On exercise, however, the heart must work harder and, therefore, the **Demands** of the myocardium increase In response to this increased demand, the **Supply** of *blood through the coronary* arteries also increases, but lags behind *(fig 23) As a result, a* slight relative anoxia of the myocardium exists for



(Figure 23)

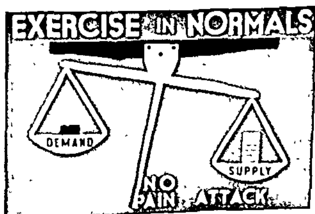


(Figure 24)

several minutes until the balance is re-established (fig 24)

With cessation of work, the opposite takes place The **Demand** is decreased to the resting level, and the coronary blood **Supply** soon decreases also to a degree sufficient for the body at rest (figs 25 and 22).

The situation is quite different in angina pectoris Here, when the Demands are increased by exercise, the coronary circulation is not able to increase (Plate 12, page 41) This discrepancy between the **Demands** for blood and the **Supply** of blood causes marked myocardial anoxia and ischemia As a result, the patient experiences heart pain This is *referred to the* chest and arms



(Figure 25)

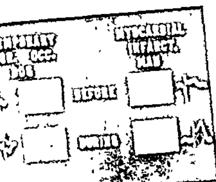


Plate 7—Electrocardiograms in coronary occlusion

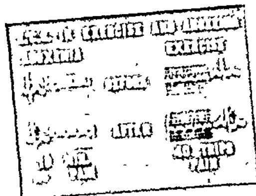


Plate 8—Similarity of electrocardiograms in exercise and anoxia.



Plate 9—Sensory nerve distribution in angina pectoris



Plate 10—The cardiac nerves





Part 3

Pathology

of

Angina Pectoris

PART 3

PATHOLOGY OF ANGINA PECTORIS

Parry's Book on angina pectoris published in 1799, includes a transcript of a letter from Dr Jenner This reads in part as follows (Plate 13, page 48) "*I was making a transverse section of the heart pretty near its base when my knife struck against something so hard and gritty as to notch it. I well remember looking up to the ceiling which was old and crumbling, conceiving that some plaster had fallen down. But on a further scrutiny the real cause appeared, the coronaries were become bony canals*" Recent work on the anatomy and pathology of the heart has confirmed the findings of Jenner

The coronary circulation is not easy to see in the gross specimen. It consists of the left coronary artery which divides into the left anterior descending branch and the left circumflex branch There is also a right coronary artery If we remove the aorta, pulmonary artery and the auricles we can see the mouths of these vessels coming off the aorta just at the base of the cusps of the aortic valves (Plate 14, page 48)

The simplest method of studying the coronary arteries is to open the lumen of each vessel with a fine pointed scissors. The objections to this method are that it may dislodge fresh occlusion and, furthermore, it is not suitable for studying vessels smaller than 1 millimeter in diameter

According to a second method, the vessels are cut across at frequent intervals and then probed This has the same drawbacks as the previous method.

Hearts have been injected with various radio-opaque materials and studied by X-Ray The vessels appear to cross each other, however, and adequate study is difficult

A simple practical method which corrects these difficulties was devised by Schlesinger in 1939 This method combines the use of Injection, X Ray, and Dissection By means of it we can study the entire course of each blood vessel down to 40 micra Areas of obstruction or narrowing too small to be seen by other methods of study are readily observed, and the presence of anastomotic circulation is easily recognized. This method has given us considerable information about the pathology of angina pectoris The method can be used in the routine examination of all post-mortem material The technique is simple

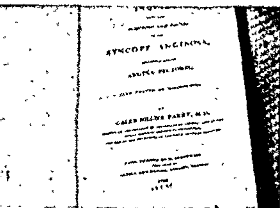
Glass cannulae, connected to short lengths of rubber tubing and metal adapters, are carefully inserted into the mouths of the coronary arteries and tied into place securely (Plate 15, page 48)

A bath of warm isotonic saline is prepared by mixing one part of 18% salt solution with 19 parts of hot tap water (Plate 16, page 48) A heating unit and thermostat adjusts the temperature of this bath to 45 degrees centigrade The heart is suspended in the bath, and a thermometer is inserted into one ventricle

While the heart is being warmed, the injection mass can be prepared (Plate 17, page 48). One and one-half grams of agar-agar are weighed out, using an ordinary balance This is placed in a pyrex container which can be covered To this is added 100 cubic centimeters of a 30 per cent solution of lead acetate in water 70 cubic centimeters of a 12 per cent solution of anhydrous sodium phosphate is added and mixed by shaking. This results in the precipitation of the insoluble and radio-opaque lead phosphate Phenolsulfonphthalein is added as an indicator With a graduated pipette, enough ten percent sodium hydroxide is added, with shaking, to neutralize until the first pink color appears Finally, distilled water is added to bring the total volume up to 200 cc Usually, 10 to 20 cubic centimeters are required The agar-agar is brought into solution by heating over an open flame Boiling is continued for 10 minutes The mixture froths vigorously, and frequent shaking is necessary to prevent its boiling over The hot injection mass is then filtered through a double layer of gauze into narrow mouthed 50 c c centrifuge tubes Finally, the injection mass is colored by the addition of dyes fuchsin is added to give a red color and methylene blue for blue The tubes are stoppered and shaken thoroughly to ensure mixing

A flask weighted with lead is filled with saline from the bath The injection mass is kept warm to prevent solidification of the agar Both cannulated arteries are connected to the flask which in turn is connected to a mercury manometer With a cautery bulb the pressure in the system is raised to 200 millimeters of mercury This forces saline into the arteries and removes the residual air and blood (Plate 18, page 48)

The containers of colored, radio-opaque mass are now fitted with two hole rubber stoppers so that pressure can be applied above the fluid and the mass forced out The containers are connected to the



SYNCOPE ANGINOSA

Plate 13—Syncope Anginosa-Parry



Plate 14—The mouths of the coronary arteries



Plate 15—The cannulae in the coronary arteries

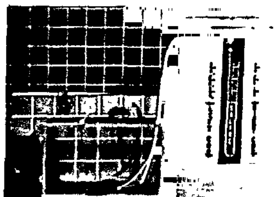


Plate 16—Apparatus for injecting hearts

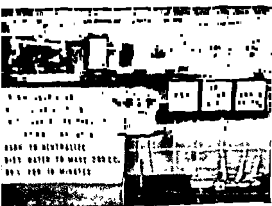


Plate 17—Preparation of injection mass.

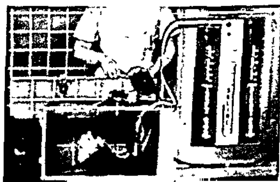


Plate 18—Washing coronary arteries with saline



Plate 19—Injecting the coronary arteries

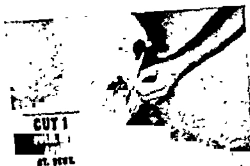


Plate 20—The first incision.

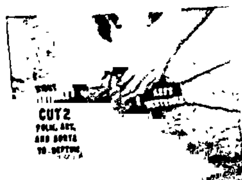


Plate 21—The second incision

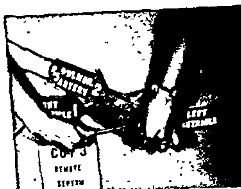
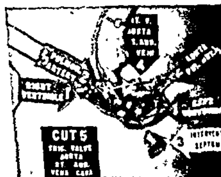


Plate 22—The third incision





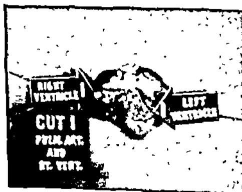
manometers, and also to the heart in such a way that the blue mass can be injected into the left coronary and the red mass into the right (Plate 19, page 49) The manometers are joined so that the pressure is the same throughout the system

The valve to the saline is shut off and the valves to the arteries turned on The pressure in the system is raised to 200 millimeters of mercury This injects the mass into both arteries simultaneously at identical pressure The red mixture fills the right coronary artery, the blue mass fills the left

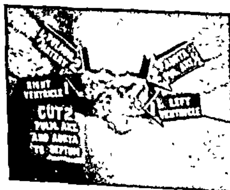
The cannulae are then clamped off, and in order to harden the agar-agar injection mass the heart is placed in a bath of ice and salt solution for about 10 minutes

The injected heart is then unrolled by a series of incisions, so that the vessels lie in one plane

The first incision starts in the pulmonary artery and divides the pulmonary valve (Plate 20, page 49) It also cuts the right ventricle to the apex just to the right of the interventricular septum This results in the opened right ventricle lying to your left,



(Figure 26)



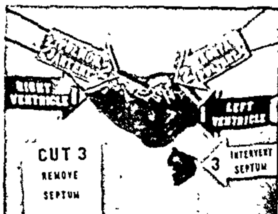
(Figure 27)

while the left ventricle remains intact (fig 26)

The second incision starts in the aorta between the two cannulated vessels and extends into the base of the interventricular septum (Plate 21, page 49) This again divides the pulmonary artery (fig 27)

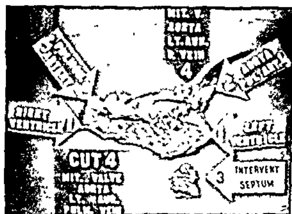
The interventricular septum is now removed in one piece as close as possible to the walls of the left ventricle

(Plate 22, page 49). A scalpel is best for this procedure This partially opens the left ventricle (fig. 28).



(Figure 28)

The fourth incision starts in one of the pulmonary veins It cuts the aortic ring and the anterior cusp of the mitral valve (Plate 23, page 49) This unrolls the left ventricle. (fig 29)



(Figure 29)

The fifth and final incision goes through the su- vena cava, the aortic ring again and the tricuspid valve (Plate 24, page 49) This permits the right ventricle to be unrolled completely (Plate 25, page 55)

The heart has now been opened without having cut any of the major vessels Furthermore, all the vessels now lie in one plane An X-ray of the unrolled heart is now taken Using the X-ray as a guide, the vessels are carefully dissected By studying the X-ray together with the dissection, tiny areas of narrowing and obstruction not recognizable by other methods of study can be detected readily

From the X-ray and the dissection, a colored diagram is made. This diagram, together with the X-ray, are permanent records (Plate 26, page 55) The myocardial tissue can be examined microscopically and then discarded.

From a careful study of the X-ray, the dissection and the diagram, the course of the coronary circulation during life can be visualized

THE NORMAL CIRCULATION

Plate 27, page 55, shows a normal heart obtained from a man 63 years of age with no cardiac disease who died of intestinal obstruction following operation In the normal heart, the injection mass flows smoothly through the entire length of the blood vessels both left and right. There is no obstruction, delay, or any evidence of collateral or anastomotic circulation This normal pattern is quite different from those seen in coronary artery disease

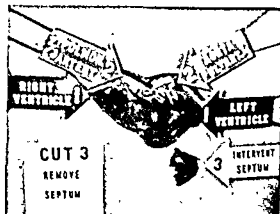
MYOCARDIAL INFARCTION

The heart shown in plate 28, page 55, was obtained from a man 50 years of age with angina for only 11 days before the onset of myocardial infarction which resulted in death The left anterior descending artery shows an area of narrowing but no obstruction near its origin The left circumflex shows a similar area of arteriosclerosis with a thrombus completely occluding its lumen The right coronary artery and distal portion of the left descending are normal The left circumflex, however, fills poorly beyond the occlusion There are no adequate anastomoses As a result, the circulation was unable to compensate for the sudden occlusion of the left circumflex artery An infarct developed in the area nourished by this vessel, and the patient died of a ruptured myocardium

ANGINA PECTORIS—One Pattern

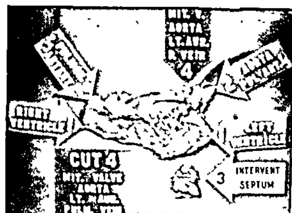
A third heart (Plate 29, page 55) was obtained from a woman 60 years of age who had angina pectoris for 5 years. At post mortem it showed one of the many patterns possible in this condition There was an old complete occlusion of the right coronary artery near its mouth This prevented the red mass from going further There was marked arteriosclerosis of the left circumflex and left descending arteries resulting in narrowed tortuous lumens, and there was a second old occlusion in the left descending artery In response to these narrowings and occlusions a rich anastomotic network was built up during life so that the circulation to the right side of the heart was maintained from the left coronary artery This anastomotic circulation was adequate to maintain life for several years despite occlusions of two of the main coronary arteries The coronary circulation was impaired, however, so that the myocardium suffered from ischemia, and the patient experienced angina pectoris

(Plate 22, page 49). A scalpel is best for this procedure. This partially opens the left ventricle (fig. 28).



(Figure 28)

The fourth incision starts in one of the pulmonary veins It cuts the aortic ring and the anterior cusp of the mitral valve (Plate 23, page 49) This unrolls the left ventricle (fig. 29)

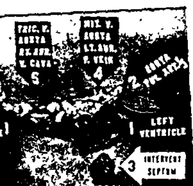


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From the X-ray and the dissection, a colored diagram is made This diagram, together with the X-ray, are permanent records (Plate 26, page 55) The myocardial tissue can be examined microscopically and then discarded



25—The fifth incision completed.



Plate 26—The complete preparation.



Plate 27—Normal circulation

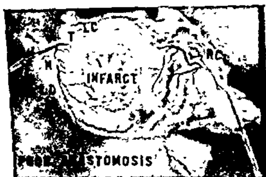


Plate 28—Myocardial infarction.

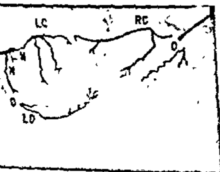


Plate 29—Angina pectoris



Plate 30—Angina pectoris

ANGINA PECTORIS—Many Old Occlusions

A fourth heart (Plate 30, page 55) obtained from a man 53 years of age, shows how extensive occlusions and anastomoses can be. The left circumflex artery had an old occlusion near its mouth. The first branches of the right coronary and the left descending arteries were also occluded There was an old occlusion of the left descending artery and also of its second major branch A fresh thrombus, the cause of death, was found in the right coronary artery and there was an old occlusion near this point Anastomoses had developed beyond these occlusions so that the branches received blood from the major vessels A seventh occlusion failed to stop the circulation The flow of blood must have been reversed in some of the terminal branches, for they received blood from the opposite sides of the heart An eighth occlusion, in a branch of the left descending also caused reversal of the flow of blood *A ninth occlusion was found in another terminal branch The* remainder of the vessels were filled by mass from both the right and left coronary arteries Despite these 9 old occlusions, this patient with angina pectoris was active in business up to 22 hours before death. There was only one, small, old infarction

An analysis of the variations in the circulatory pattern found in coronary artery disease shows the following

First, complete occlusion of one or more of the major vessels is a common occurrence

Second, these occlusions do not prevent longevity and physical activity, providing an adequate collateral circulation develops

Third, angina pectoris occurs when following coronary occlusion the collateral circulation is not *adequate to Supply the Demands of the* myocardium during exercise

Part 4

Treatment of Angina Pectoris

PART 4

TREATMENT OF ANGINA PECTORIS

A tremendous number of methods of treatment have been advocated for angina pectoris (Plate 31, page 61) According to the published reports, each of these methods makes the patient more comfortable Little exact or comparative information of their effect has been available, however

Since, so far as we know, angina pectoris exists only in humans the benefits of therapy must be evaluated not in laboratory animals but in patients.

The value of any method of treatment can be measured objectively by the Standardized Exercise Tolerance Test For example, the value of nitroglycerin can be demonstrated as follows The amount of work which a patient can do without medication before developing pain is found by repeated tests under the usual standardized conditions of cold, food, and so forth which have been found to give constant results The patient then places a tablet under his tongue and waits a measured length of time for it to take effect The test is then repeated. By means of a counter and stop-watch, we again measure the amount of work which he can do By this method we can demonstrate that most patients after nitroglycerin can do more work, without pain, than was possible without medication

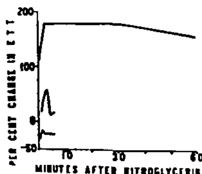
Obviously, **since most patients with angina develop their attacks on exertion, any treatment of value must enable the patient to perform more work before developing pain.** This objective method of study makes it possible to evaluate the effect of treatment without being influenced by the impression of the patient or the physician

This objective method of study has been used in the Angina Clinic of the Beth Israel Hospital of Boston since 1932, the results have proven of value in the clinical management of patients Up to the present time the efficacy of over 70 different methods of treatment have been tested and compared (Plate 32, page 61)

A group of about 20 methods have been found of great value, others have been of slight or only moderate value, while despite the claims of the manufacturers, many are of little value

THE NITRITES

Generations of patients and doctors can testify to the value of nitroglycerin for decreasing the length of attacks It is also helpful in preventing attacks For example, studies in one patient (fig 30, center curve) showed him able to do 20 per cent more work if exercise is started 1 minute after nitroglycerin After 2 minutes he can do 40 per cent more work After 3 minutes, 60 per cent more Shortly after this, however, the effect diminishes rapidly and disappears within 10 minutes Experience shows that this is a moderate response to nitroglycerin



(Figure 30)

Other patients show far more striking benefit They can do at least 100 per cent more work for at least one half hour after taking nitroglycerin (fig 30, top curve) A third group of patients show no response to this therapy (fig 30, bottom curve)

The response to nitroglycerin can be used to determine the probability of a patient's response to other forms of treatment (Plate 35, page 61)

For example, one group of patients is able to perform approximately 100 per cent more work when given nitroglycerin When given other drugs, they show a definite increase in exercise tolerance and, more important, practically complete disappearance of attacks in daily life This group consists of a little less than one-third of the patients

A second group is able to do approximately 50 per cent more work after nitroglycerin When these patients are given other medication they show a definite decrease in frequency of attacks in daily life but attacks are not likely to disappear entirely This group includes one third of the patients

A third group is unfortunate for they show no response to nitroglycerin or any of the usual methods of treatment This group includes 40 per cent of patients

It is thus possible to divide patients with angina pectoris into three groups on the basis of their response to therapy

That these results are due to actual changes in the heart and not to psychological influence can be shown by taking electrocardiograms during and following exercise (Plate 36, page 61) Under ordi-

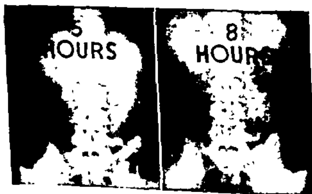
THE PURINES

According to animal experiments and electrocardiographic studies in patients, these drugs are vasodilators Theobromine and theophylline which are obtained from cocoa or tea are relatively insoluable They can, however, be joined with many other substances to form loose chemical combinations which increase their solubility and effectiveness Many of these have been prepared and are available for use The comparative value of 18 different xanthine derivatives have been measured and demonstrated by the Standardized Exercise Tolerance Test A few are of considerable value Many widely advertised brands, however, are of little value

In order to control these studies, and to eliminate the psychological factor, the response has been compared to that obtained following sodium bicarbonate or lactose prepared to resemble the medication being studied (Plate 34, page 61) The response to medication when disguised by dipping in tincture cudbear served as an additional control Electrocardiographic studies further differentiate between active and inert therapy

The most effective of these xanthines are Theobromine Sodium Acetate, Theophylline Sodium Acetate, and Theophylline Calcium Salicylate (Plate 37, page 72)

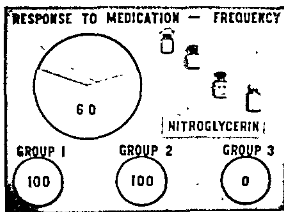
All the xanthines when given in sufficient dosage cause gastric distress but this can be prevented by encasing them in an enteric coating which enables them to pass through the stomach intact and disintegrate in the small intestine (Plate 38, page 72) In most patients absorption takes place in 4 to 6 hours (fig 33) It must be remembered therefore, that it is the tablet given before retiring which is effective in the early morning



(Figure 33)

nary conditions, the patient illustrated shows definite changes in the S-T segment of the electrocardiogram at the time when an attack is precipitated by exercise When nitroglycerin or other effective vasodilators are given, the patient can perform a greater amount of work than is otherwise possible At the same time, the electrocardiographic changes ordinarily observed either do not occur or are markedly diminished

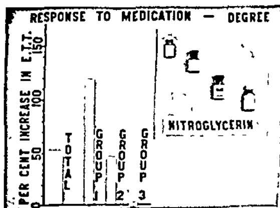
Nitroglycerin can be absorbed readily by the mucous membrane under the tongue Since the duration of attacks is short, the medication must be absorbed within a period of a few seconds For this reason, the more soluble hypodermic tablets are preferable to tablet triturates A properly prepared tablet should dissolve in water at room temperature in about 1 minute The toxic or untoward effects of nitroglycerin are seen more commonly in young individuals than in those with arteriosclerosis They are due to vasodilation and consist of a burning and flushing of the face, together with headache, and occasionally faintness, if there is a marked fall in blood pressure In most instances 1/200 grain of nitroglycerin is as effective as larger doses and is less likely to give untoward effects



(Figure 31)

Nitroglycerin is one of our most valuable drugs (figs 31, 32) It is clinically effective in 60 per cent of patients (Groups 1 and 2) It is of low toxicity and low in cost Its use before work results in an average increase in exercise tolerance of about 120 per cent in patients of Group 1 The optimum dose is 1 200 grain, but 1/400 every hour is of considerable value in some patients

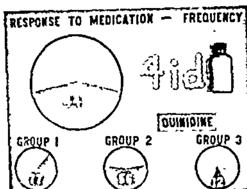
Amyl Nitrite can be used by inhalation It is an effective nitrite but offers no particular advantage over nitro glycerin and is more costly The same is true of Octyl Nitrite which also is given by inhalation Erythol-tetranitrate and Mannitol Hexanitrate are considerably less effective Sodium Nitrite is of little or no value



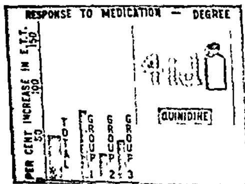
(Figure 32)

QUINIDINE SULFATE

Quinidine has not been used sufficiently in angina pectoris Experience in over 100 patients has shown that this drug is clinically effective, low in toxicity in patients with angina, and low in cost The administration of the sulfate in doses of 5 grains four times a day helps approximately 44 per cent of patients (figs 36-37) What is more important is that it is often of value in patients not benefitted by the xanthines Its use results in an increase in exercise tolerance of about 50 per cent



(Figure 35)



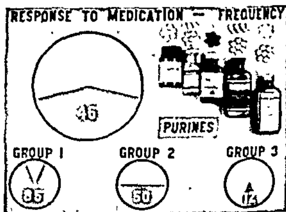
(Figure 37)

Objective evidence of the value of quinidine can be obtained from the study of the electrocardiogram during work (Plate 41, page 72) After quinidine is given, exercise fails to induce the S-T segment changes usually seen when no medication is administered Quinidine also causes a prolongation of the Q-T interval (the duration from the beginning of the QRS to the end of T) This specific effect shows that quinidine acts for about 6 hours after administration

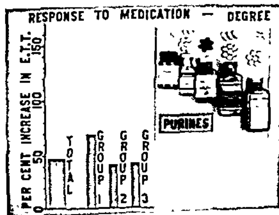
The efficacy of these preparations can be illustrated by comparing them with nitroglycerin (Plate 39, page 72). Administration of nitroglycerin before exercise enables patient, S W., to do over 75 percent more work than is otherwise possible This response reaches its maximum in 3 minutes and is gone in 20 minutes.

The effect of Theobromine Sodium Acetate is equally striking, although somewhat less pronounced Fifteen minutes after the administration of a capsule or a tablet, this patient can do 57 per cent more work than was possible without medication This effect persists for about 3 hours

The therapeutic action of the enteric coated tablet is more delayed and also more prolonged The enteric coating prevents absorption for about 4 to 6 hours, but its therapeutic effect continues for almost 10 hours Following its use, this patient is able to do about 50 per cent more work than was otherwise possible



(Figure 34)

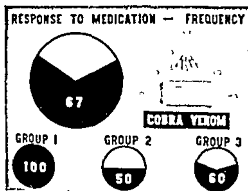


(Figure 35)

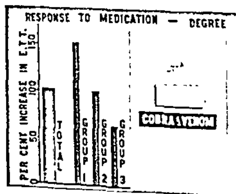
The purines are of value in about 45 per cent of the total number of patients (figs 34, 35). 85 per cent of those in group 1, but only 12 per cent in group 3 The average increase in exercise tolerance following the use of the purines is approximately 50 per cent

COBRA VENOM

The use of cobra venom as an analgesic deserves special mention. Many patients to whom the drug is given develop a marked decrease in attacks of pain in daily life and are able to do considerably more work under standardized conditions. Furthermore, this drug is of value in many patients who do not respond to other available medications (figs. 41, 42).



(Figure 41)



(Figure 42)

There are two drawbacks to its general use. First, the drug must be injected intramuscularly every day for at least a week before an effect is obtained, and it must be continued indefinitely at least twice weekly to maintain an effect. Many patients are unable to continue this form of treatment because of inconvenience and cost. The second drawback is that it acts solely as an analgesic. The lack of vasodilating action can be shown by electrocardiographic studies (Plate 42, page 72). If we compare the electrocardiogram with the patient standing at rest with that taken during an attack, we see the typical S-T deviation. Cobra venom causes no change in the electrocradiogram at rest. After exertion, however, although the patient does *not* experience pain, the electrocardiogram is still typical of angina.

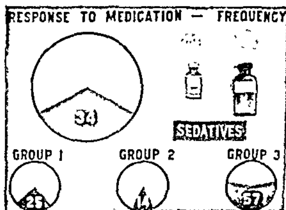
Cobra venom is of value for symptomatic relief even though there is no basic cardiac improvement. In selected cases it may obviate the necessity for surgery.

SEDATIVES

The sedatives have a definite place in the treatment of angina pectoris, both alone and in combination with other drugs. Many such combinations are available on the market (fig. 38).



(Figure 38)

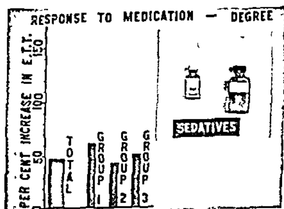


(Figure 39)

The beneficial results of the sedatives are probably due to a decrease in emotional activity which results in a decreased frequency of attacks. As many patients state "it makes the pain easier to bear." The sedatives most commonly used are the bromides and the barbiturates, especially phenobarbital.

The opiates are extremely valuable. Since they are habit forming, they are best reserved for acute exacerbations of the disease.

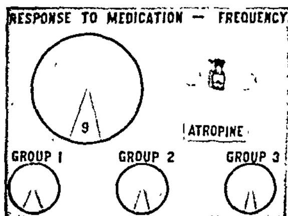
An increase in exercise tolerance following the use of these drugs is not common, but does occur in some patients who are helped by few other drugs (figs. 39, 40).



(Figure 40)

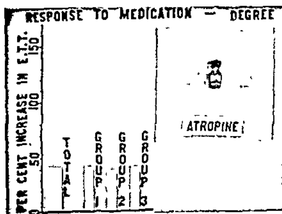
THERAPY ACTING ON THE AUTONOMIC NERVOUS SYSTEM

Therapy which acts on the autonomic nervous system includes atropine, physostigmine, and carotid sinus stimulation All these are of some value in angina



(Figure 43)

Only a few patients respond to atropine, but this drug is of importance because it is of benefit to some persons in group 3 (figs 43-44)



(Figure 44)

Carotid sinus pressure and physostigmine, both of which stimulate the vagus nerve also have a beneficial effect in some patients

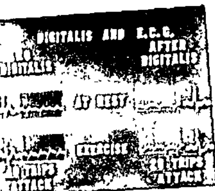


Plate 43—Digitalis may be injurious in angina pectoris

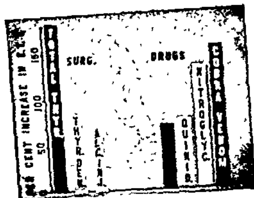


Plate 44—Comparative value of medical and surgical procedures

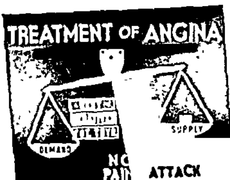


Plate 45—Methods of decreasing demand in angina pectoris

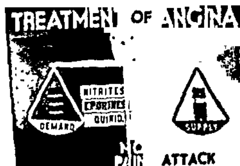


Plate 46—Methods of increasing supply in angina pectoris

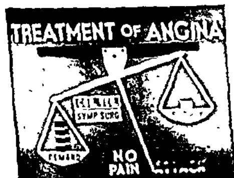


Plate 47—Symptomatic relief without

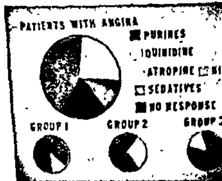


Plate 48—The "routine" treatment of

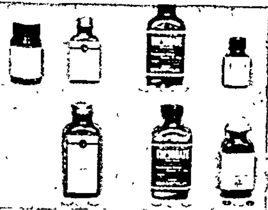
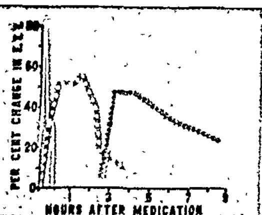


Plate 37 —The most effective purines



Plate 38 —Uncoated and enteric coated purines



ate 39 —The value of theobromine sodium acetate



Plate 40 —Enteric coated potassium iodide

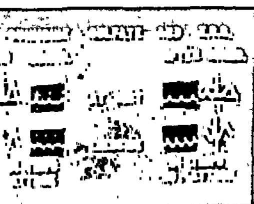


Plate 41 —Value of quinidine sulphate in angina pectoris

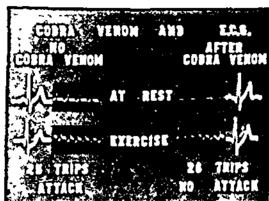
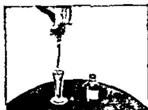


Plate 42 —Symptomatic value of cobra venom

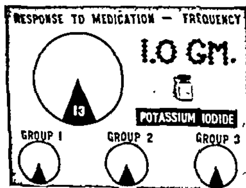
IODIDES

The iodides are of undoubted value in those few cases of angina secondary to thyrotoxicosis or syphilis The doses commonly used, however, have proven of little value in the usual case of angina pectoris It frequently requires as much as 35 drops of the saturated solution to equal 1 c c or 1 gram of drug, hence 15 drops 3 times a day is really a small dose (fig 45)



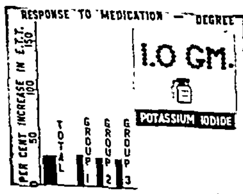
(Figure 45)

Larger doses, one gram 3 or 4 times daily, have been found to be of greater value in a few cases, especially those in Group 3 who are



(Figure 46)

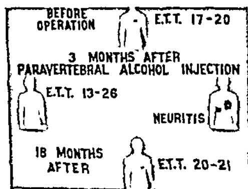
unlikely to be helped by other medication In order to avoid gastric distress, this is best given as an enteric coated tablet (figs 46-47), (Plate 40, page 72)



(Figure 47)

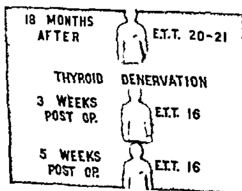
half refused operation when the risks and likelihood of benefit were explained to them As a net result, surgery was performed in only seven per cent of the patients with angina who came to the hospital for treatment

The results in patient R S who was incapacitated by angina pectoris for over 6 years illustrate the effect of surgery on the sympathetic nervous system (fig 50) His exercise tolerance was 17 to 20 trips, and his pain was experienced in the second interspace on the left and along the inner aspect of the left arm After paravertebral alcohol



(Figure 50)

injection the *severity of the attacks* decreased strikingly They were limited to a small area encircling the left wrist His ability to work, however, was not increased Simultaneously, he developed a severe neuritis which persisted for over a year Before the neuritis subsided completely the anginal syndrome returned in its original location and severity Eighteen months after (fig 51) because of continuation of his



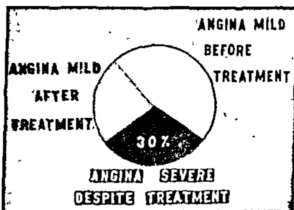
(Figure 51)

DIGITALIS

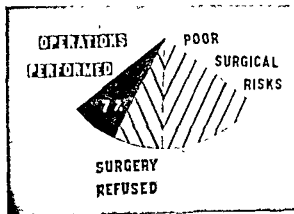
The digitalis bodies deserve special mention, for these drugs are frequently harmful to patients with angina pectoris This can be shown *objectively* by electrocardiograms and exercise tolerance studies For example, when no medication was given, patient M L developed an attack on performing 40 trips, and the *eletrocardiogram shows the* typical S-T depression (Plate 43, page 73) When these studies were repeated after digitalization, the electrocardiogram taken at rest showed no striking changes On exercising, however, the patient developed a severe attack after 28 trips The electrocardiogram shows a marked depression of the S-T *segment This striking decrease in exercise* tolerance was accompanied clinically by a corresponding increase in the frequency and severity of attacks in daily life

SURGERY

There are numerous reports in the literature on the surgical treatment of angina pectoris Obviously, evaluation of the results of surgery is just as unreliable as the purely clinical evaluation of medical therapy Experience at the Beth Israel Hospital indicates that in 46 per cent of patients with angina, the symptoms even without treatment are so mild that surgery is not indicated (fig 48) In another 24 per cent, the response to medical treatment makes surgery unnecessary This leaves 30 per cent of patients in whom the symptoms were severe despite therapy (fig 49) Since angina pectoris is the *result of an aging process,* it is not surprising that one-half of these are poor surgical risks Of the remainder,



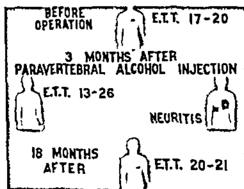
(Figure 48)



(Figure 49)

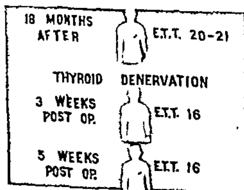
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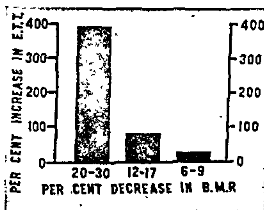


(Figure 51)

angina, thyroid denervation was *performed* He was *more comfortable* after this operation His exercise tolerance remained the same, but the pain was less severe and was limited to the left arm Five weeks later, however, the symptoms returned

Sympathetic nerve surgery, therefore, results in symptomatic relief without any increase in ability to work

The most striking results which we have observed were obtained by Total Thyroidectomy (fig 52) This procedure lowers the metabolism and thereby decreases the work of the heart Those patients whose basal metabolic rates were decreased by 20 to 30 per cent showed an average increase in exercise tolerance of nearly 400 per cent *They were also practically free* from attacks in daily life



(Figure 52)

Patients who showed a moderate fall in basal metabolism showed proportional improvement, while those who showed little change in metabolism had no clinical improvement and no appreciable increase in exercise tolerance

We have seen that few patients require surgery In suitable cases, however, the beneficial response to total thyroidectomy may be marked and the untoward effects negligible

The comparative effect of medical and surgical therapy can be illustrated as follows (Plate 44, page 73) Following the use of the purines, patients in Group I show an increase in *exercise tolerance* of 70 per cent The use of quinidine results in a 75 per cent increase in *exercise tolerance* Nitroglycerin causes an increase of 120 per cent Cobra venom enables patients to do about 150 per cent more work Although the patient may be more comfortable following paravertebral alcohol injection, there is usually no appreciable increase in ability to work The same is true following denervation of the thyroid gland After total thyroidectomy on the other hand, the average increase in exercise tolerance of all patients studied was 180 per cent

THE ROUTINE TREATMENT OF ANGINA PECTORIS

Treatment must be individualized Specific emotional or exertional factors responsible for attacks can be eliminated in many cases. In others hyperthyroidism, anemia or arrythmias may be corrected with benefit In most patients, medication is necessary. As a result of measuring the value of 70 different therapeutic agents, it is possible to present a practical plan of therapy using those preparations most likely to be of value in the largest number of cases (Plate 48, page 73)

The purines, if given in adequate doses, are most likely to be of benefit These will help about 43 per cent of all patients, 85 per cent of Group 1, 50 per cent of Group 2, but only 12 per cent of Group 3 **If medication is of value, it will be evidenced within a week by complete or almost complete disappearance of attacks in daily life. Any improvement short of this is not due to the medication used.** If no satisfactory response is obtained, it is advisable to administer quinidine sulfate as the next drug most likely to be of benefit Sixty per cent of all patients are helped by either the purines or quinidine This includes all patients in Group 1, 70 per cent of Group 2, but only 24 per cent of Group 3 The treatment of the remaining 40 per cent of patients, especially those in Group 3, is the present problem and recent studies have concentrated on a search for therapeutic agents which will help this group Atropine sulfate, though not as valuable as the purines or quinidine, may help 10 per cent of such patients in Groups 2 and 3 The sedatives, phenobarbital or codeine sulphate, will help other patients in Group 3 and, of course, are of value as an adjunct to other therapy in all patients There remains about 20 per cent of patients, two-thirds of whom are in Group 3 who respond to none of these medications

In patients with severe angina, it is advisable to use combinations of medication Such treatment should begin with a period of complete bed rest Adequate sedatives, either barbiturates or opiates, should be given This may be combined with small frequent doses of nitroglycerin 1 400 every hour In some instances, it may be advisable to give an active purine in addition, and, if necessary, quinidine sulphate.

This regime will result, usually, in complete disappearance of pain, unless there has been a recent coronary occlusion or there is a strong emotional element Cobra venom is advisable if the symptoms return when the patient is allowed up and about again If this fails to give relief, surgery should be considered, total thyroidectomy in suitable cases, sympathetic nerve surgery in others

In summary, since angina pectoris occurs when the Supply of blood is inadequate for the Demands of the heart, treatment may take 3 general forms.

The first consists of **decreasing the Demand** for blood by rest, sedatives, or if necessary, decreasing the metabolic needs of the body by total thyroidectomy (Plate 45, page 73).

The second method of treatment consists of **increasing the supply** of blood by the use of drugs such as nitroglycerin, the purines, and quinidine sulphate (Plate 46, page 73)

The third type of therapy (Cobra Venom and Sympathetic Surgery) should be used only where it is impossible to readjust the balance between Supply and Demand by the above methods (plate 47, page 73) Here, the underlying physiological mechanism responsible for the attacks is not altered, but the procedure **interrupts the sensory nerves** so that pain is not experienced.

The aims of treatment in angina pectoris are First, relief of discomfort, *Second, prolongation of life and prevention of myocardial infarction*, and, Third, economic rehabilitation if possible, or at least prevention of cardiac invalidism The plan of treatment just outlined, together with a thorough understanding of the patient and his condition, goes a long way towards fulfilling these aims